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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

Lead Encephalopathy: Treatment and Recovery. Report of Case.
Israel Binder, M.D. 51

Poliomyelitis: Infectious or Metabolic?
W. J. McCormack, M.D. 58

Pseudohermaphroditism in Twins.
Martin L. Stone, M.D., and Adrian Zorgniotti, M.D. 74

Atypical Pyloric Stenosis. Report of Two Cases in Negro Infants in Whom the Vomiting Began on the First Day of Life.
Althea D. Kessler, M.D., and Roland B. Scott, M.D. 78

The Relationship of Trauma to Sickle Cell Anemia. Report of Case.
Max Kliger, M.D. 82

Pediatrics Half a Century Ago: Acute Myocardial Insufficiency in Some Infections in Children.
F. Forchheimer, M.D. 84

Clinical-Pathological Conference, Willard Parker Hospital. 91

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CONTENTS

ORIGINAL COMMUNICATIONS

Lead Encephalopathy: Treatment and Recovery. Report of Case.
ISRAEL BINDER, M.D. 51

Poliomyelitis: Infectious or Metabolic?
W. J. MCCORMICK, M.D. 56

Pseudohermaphroditism in Twins.
MARTIN L. STONE, M.D., and ADRIAN ZORGNIOTTI, M.D. 74

Atypical Pyloric Stenosis. Report of Two Cases in Negro Infants in Whom the Vomiting Began on the First Day of Life.
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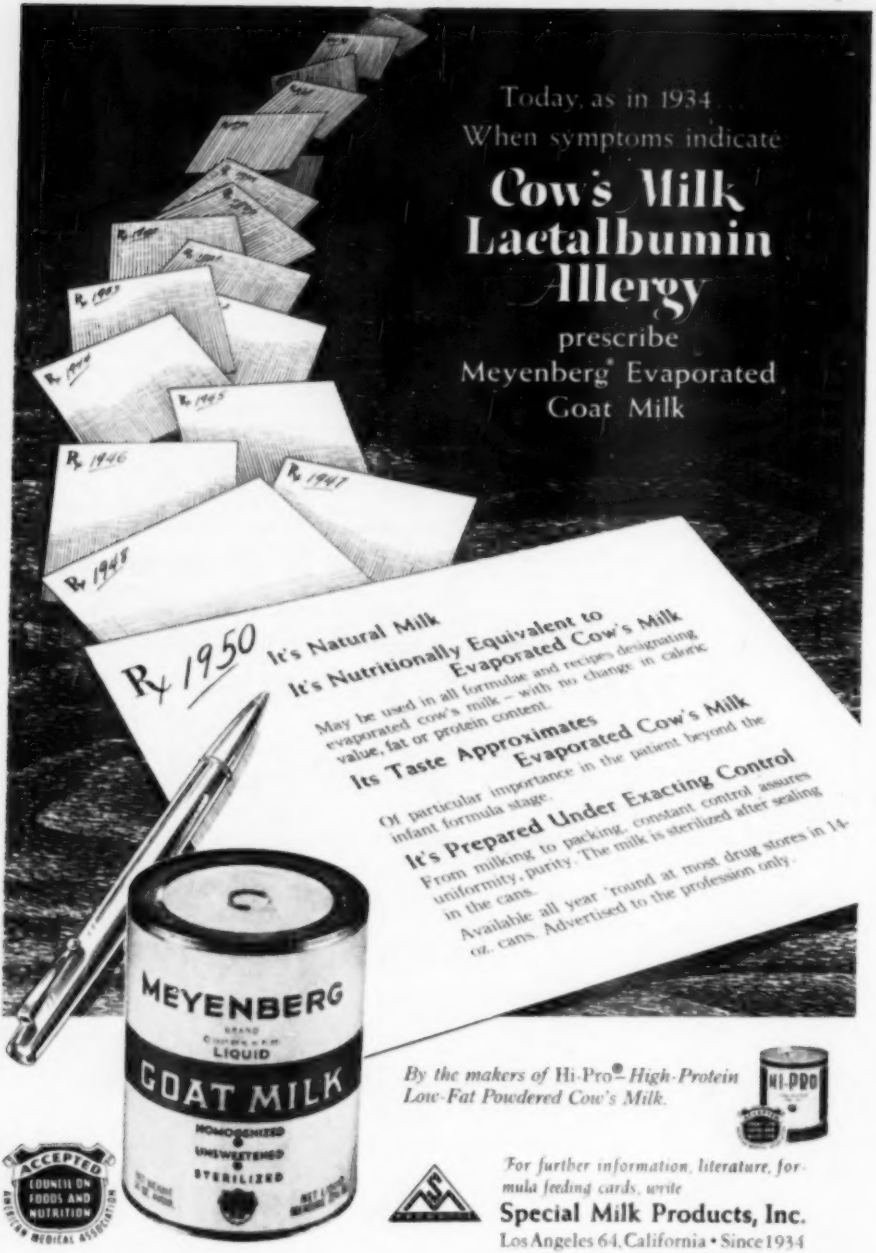
The Relationship of Trauma to Sickle Cell Anemia. Report of Case.
MAX KLIGER, M.D. 82

(Continued on page 5)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(Continued from page 3)

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Acute Myocardial Insufficiency in Some Infections in Children.
F. FORCHHEIMER, M.D. 84

CLINICAL PATHOLOGICAL CONFERENCE

Willard Parker Hospital, Meeting Held September 20, 1949..... 94

ITEMS

Soy Bean Milk in Feeding of Infants..... 77

Complicated Visceral Abnormalities..... 77

Streptomycin in Tuberculous Meningitis..... 81

Complications of Herpes Zoster..... 93

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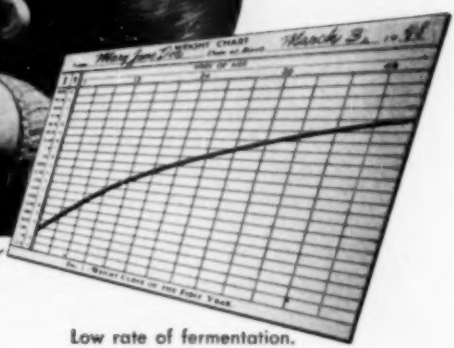

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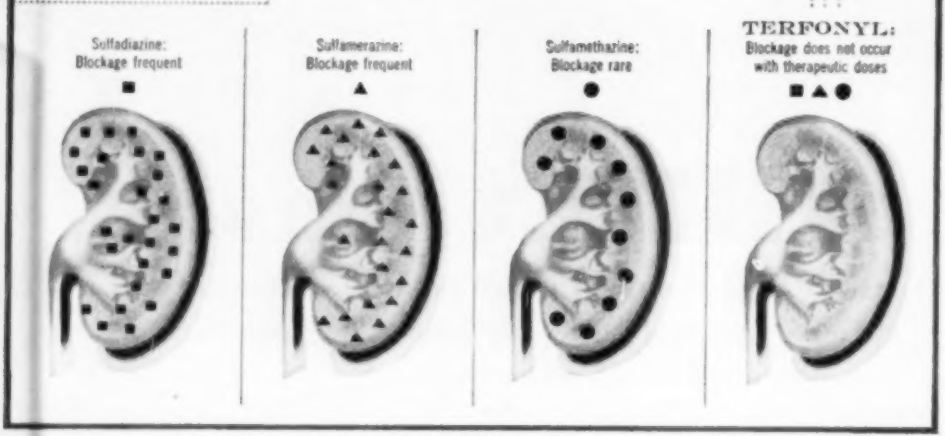

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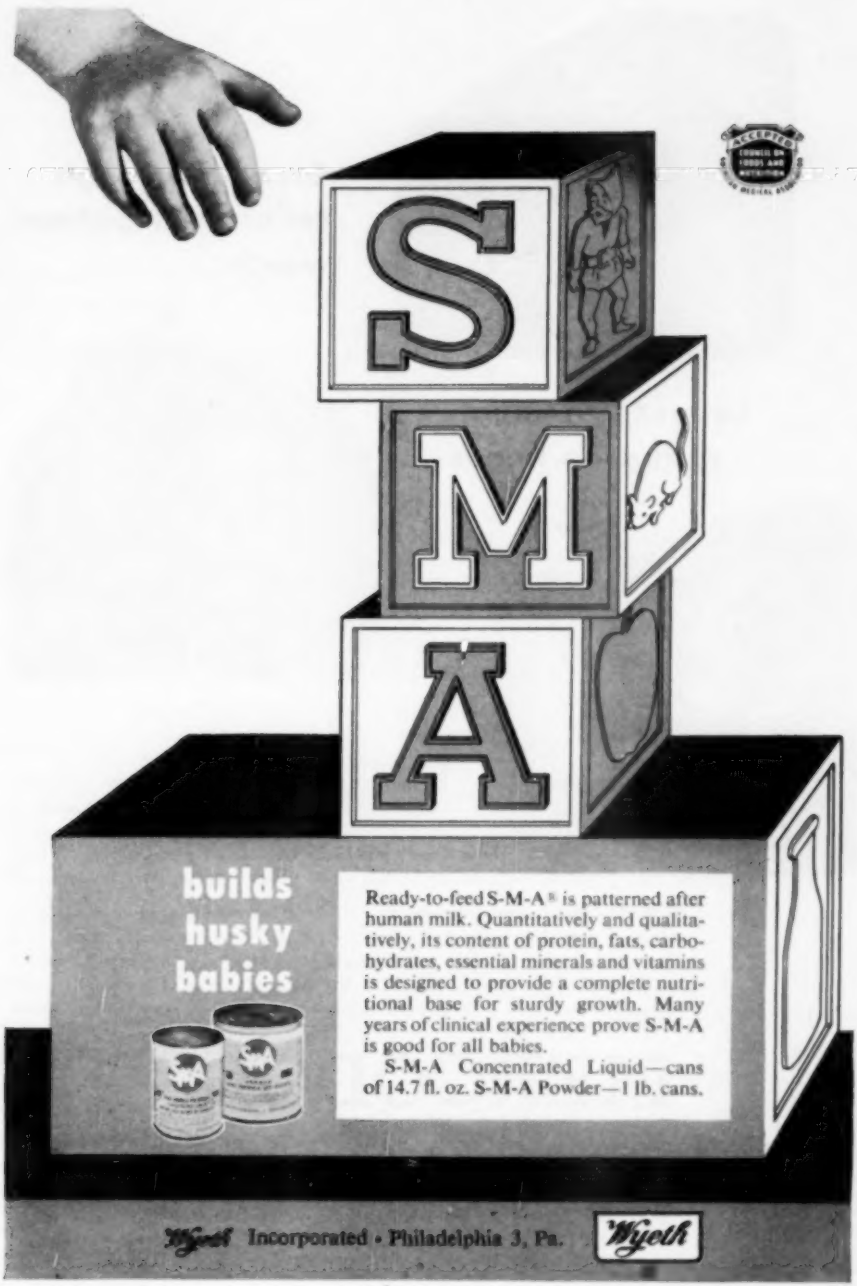
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VOL. 67 FEBRUARY 1950 No. 2

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LEAD ENCEPHALOPATHY: TREATMENT AND RECOVERY

REPORT OF CASE

ISRAEL BINDER, M.D.

Philadelphia

The incidence of lead poisoning in children in recent years has markedly decreased, but occasionally a case may appear caused by an unusual source of lead. The drop in the incidence of lead poisoning may be attributed to the recognition of the danger of the use of lead in objects easily obtainable by children, and to its elimination from household utilities.

That the source of lead in cases of poisoning is varied is shown in the following examples. In the past, cases have been reported which have been traced to the use of lead plumbing in the water pipeline of the house, and also from lead-containing paints and enamels eaten by babies from the side of their cribs.[1,2] McKhann,[3] and Wilcox and Caffey[4] have noted the occurrence of lead poisoning in breast-fed children following the use of lead nipple shields and also following the application of lead acetate ointment to the breast of the mother[3]. McKhann has also called attention to the fact that children between the ages of one and three years, having the natural tendency of putting various things into their mouths, have developed lead poisoning from the continual chewing and gnawing of metal toys containing lead. Kato[5] mentions a series of cases, a percentage of which were traced to the continuous use of lead-containing face powder. In children of school age, Aik-

From the wards of the Women's Homeopathic Hospital of Philadelphia.

man[6] has pointed out the occurrence of lead poisoning following the inhalation of dust from colored chalks.

Although today other metals for the most part have been substituted for lead, the occurrence of lead poisoning, though low, must still be kept in mind. Surveys of the literature continually emphasize the importance of early diagnosis in lead poisoning, giving as early symptoms: restlessness, easy excitability, pallor of the skin, anorexia, constipation, headache and colic. Since these symptoms are so common in children in many other conditions, the diagnosis of lead poisoning is readily obscured and actually can only be recognized clinically after the onset of symptoms caused by increased intracranial pressure. This will be noted in the case described here.

CASE REPORT

H.K., a negro male, three years of age, was admitted to the hospital at 3:15 A.M. with a convulsion which lasted 45 minutes. The patient was well until the evening of his admittance to the hospital. Early in the evening he complained of a headache and later developed a convulsion which commenced five minutes before his entrance to the hospital.

His family history was negative showing no tuberculosis or epilepsy. His parents and two sisters were living and enjoying good health. His past personal history revealed that he was of normal birth, breast-fed, had measles, chickenpox, and had been inoculated for scarlet fever and diphtheria. There was no history of a previous convulsion.

Physical Examination showed the patient to be a well-nourished, well-developed negro male, three years of age. The child was in a convulsion and was incontinent, both anal and bladder contents having been emptied. He vomited four ounces of a mucoid material. Examination of the skin revealed a generalized fine papular rash. There was no adenopathy, and the lymphatics were not palpable. The eyes were rolled upward at the time of the first examination. (After two days a horizontal nystagmus was noted followed by a swelling of the upper lid on the third day). In examining the mouth and oral cavity, a pustule was noted at the corner of the left upper lip, and several pale spots were found on the mucous membrane of the right inner cheek. The lungs were

clear to percussion and auscultation. The heart was normal in size and shape. In the pulmonic area the systolic sound was obliterated by a swishing sound. There was no abnormal, palpable or ausculatory findings in the abdomen.

Upon entrance to the hospital, the patient's temperature was subnormal. On the third day the temperature rose to 102°F. and continued to rise to 103°F. on the fifth day.

Neurological Examination revealed a comatose patient who responded poorly to stimuli. His upper and lower extremities were flaccid with apparent complete lack of tone. Cranial nerve examination was negative, except for a questionable nystagmus. The pupils were round and equal and an ophthalmoscopic examination revealed no abnormalities. Deep tendon reflexes of the upper and lower extremities were absent. Abdominal reflexes were present. No pathological reflexes were noted except a positive Babinski on the left side. These findings were suggestive of an encephalomyelitis. The etiology of the condition did not seem to be bacterial in origin since there were negative spinal fluid findings. The child had repeated seizures on the second and third days after his hospitalization.

A neurological examination a few days later showed that there was a slight return of muscle tone and a disappearance of the Babinski on the left side. There was also a return of tendon reflexes, though depressed.

LABORATORY FINDINGS

1. A blood count performed on the day of entrance showed 3,400,000 red blood cells, 10,000 white blood cells and a color index of 0.75. Four days later, the red blood cells were 3,150,000 and the white blood cells 7,500, the color index being 0.77. Differential examination revealed basophilic stippling.

2. Further blood studies showed a negative serologic test for syphilis, no malarial parasites, a blood sugar of 90 mgm., blood calcium of 7.3 mgm., and blood urea of 13 mgm.

3. Urinalysis revealed nothing unusual.

4. Fecal examination for parasites was negative.

5. Spinal fluid examination showed an increased pressure, 74 mgm. of sugar, 730 mgm. of chlorides, no increase in proteins. Kolmer and Mazzini tests were negative. A smear for bacteria was negative and culture proved to be sterile.

6. A patch test was negative.

Due to the presence of basophilic stippling in the red blood cells an X-ray was taken of the long bones and a lead line was clearly discernible. This will be seen in Fig. 1.

The diagnosis of lead poisoning having been made, the mother was questioned again to determine the source of the lead. It was found that the child had been chewing on crayon for some time before his illness.

TREATMENT

The treatment of lead poisoning may follow one or the other of two principles. The first is a de-leading process, the second promotes the storage of lead in the long bones. Since this child

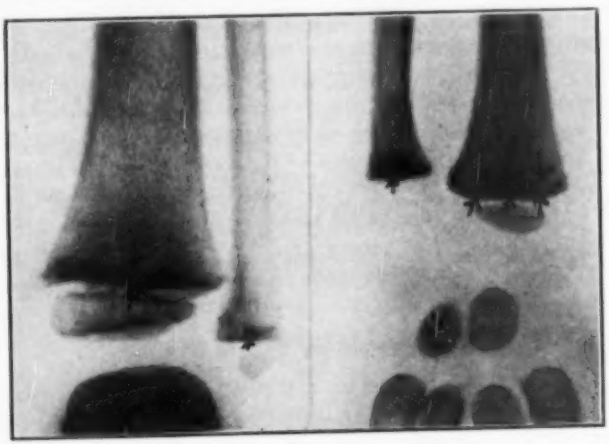

Fig. 1. X-ray of the long bones showing a lead line clearly discernible.

was in such a critical situation, it was decided to get the lead out of the blood stream as soon as possible. For this purpose, the second of the two principles was started. But before this could be carried out, the convulsions had to be controlled. Four grains of sodium phenobarbital was administered subcutaneously and also 20 grains of sodium bromide and 20 grains of chloral hydrate per rectum. This course was repeated with the onset of new convulsive movements.

In order to promote storage of lead in the bones, 1 gm. of calcium gluconate, 100 mgm. of vitamin C and 5 drops of Drisdol were given three times a day. When the elevation of temperature first appeared it was not known whether the child had a septic or a toxic condition. Because of this 7.5 grams of sulfadiazine was given for 48 hours without effect. Penicillin was then administered, 20,000 units every three hours. Following this the temperature dropped to normal, remained normal for about one week, and then climbed again to 103° F. The temperature hovered between 103° F. and 104° F. for three days. 20,000 units of penicillin was again administered every three hours for three consecutive days with no effect on the temperature. It was then decided to give sodium sulfadiazine intravenously, 2.5 Gm. being given every 12 hours. The temperature gradually declined to normal. It was later felt that the sulfadiazine and penicillin had no effect upon the temperature, but that the variations in the temperature were due to the plumbism itself.

During the course of the disease the child developed an anemia and it was necessary to transfuse 250 cc. of blood on several occasions. The patient continued to improve daily and made excellent progress. There was no indication of mental retardation or deterioration, for the child seemed quite alert upon recovery. At the end of 45 days of hospitalization the child was discharged in good physical condition.

SUMMARY

A case of lead poisoning in a three-year-old Negro male is presented. The diagnosis was obscure until basophilic stippling of the red blood cells and the lead lines, as seen in roentgenographic studies of the long bones, were noted. The child was treated according to the principle whereby the lead storage in the bones is increased. The child was discharged in good physical condition 45 days after entrance to the hospital.

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POLIOMYELITIS: INFECTIOUS OR METABOLIC?

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Since the work of Flexner and Lewis (1909) in America and almost simultaneously that of Landsteiner and Levaditi in Europe, involving the experimental production of paralytic disease in monkeys by intracerebral inoculation of an extract from the central nervous system of a victim of poliomyelitis, this disease has been generally regarded as specifically infectious and communicable. This led to the theory of the filtrable virus as the etiological agent, and to much costly and ineffectual research on the question of "means of spread," "portal of entry," immunology, prophylaxis and therapy, based on the assumption of a specific ultramicroscopic organism as the culpable agent. Convalescent serum, astringent nasal sprays, chemotherapy and quarantine have had no demonstrable effects—in fact limitation of quarantine to the febrile stage is now being advocated in some quarters. As aptly stated by Davison,[1] "isolation for three weeks of patients and contacts is required by most boards of health since 1916, but the evidence then and now does not indicate that anything is accomplished by this procedure. The disease rarely attacks more than one member of a family, and cases developed by contact are conspicuously rare. Of 2,070 persons definitely exposed, only 14 contracted the disease. Patients with the non-paralytic or subclinical type of the disease must be so common that nearly the whole population should be isolated." But the fact that the rising incidence of the disease goes on apace (the 1949 incidence in the United States, 42,375, exceeds all previous records) shows that there is something radically wrong with our approach to this problem.

It is presently customary to refer to subjects of this disease as being stricken, smitten or attacked, as for instance in the caption "When Polio Strikes." It is human nature to seek external causes for disease. It is easy to make the fly or the mosquito the scapegoat. Solomon has said: "The curse causeless shall not come." The principle of curing and preventing disease by dependence upon drugs and vaccines is physiologically unsound. Disease is caused by abnormal conditions of life, and, unless correction is made in this respect, no radical therapeutic and prophylactic relief can be

expected. We naturally look for short cuts, but "the longest way round is often the shortest way home." The day of miracles and magic has passed.

Following the 1937 outbreak of poliomyelitis in Toronto the author made an extensive study of the comparable features of beriberi and poliomyelitis, and advanced the hypothesis of vitamin B deficiency as a basic etiological factor in the latter disease.[2] Subsequently, the author published other papers[3-7] in support of this hypothesis.

The historical background of these two diseases is strikingly similar. Beriberi has been recognized in the densely populated countries of the Orient for many centuries. Its incidence in epidemic proportion, however, has become more noticeable following the introduction of the practice of rice polishing, whereby the vitamin B factors are removed from the grain. Likewise poliomyelitis, although apparently known to have occurred sporadically since ancient Egyptian times, did not appear in epidemic form until 1840, when it was reported by Heine in Germany, immediately following the adoption of the patented process of white-flour milling (Vienna, 1839), by which the vitamin-B-containing elements, the bran and the germ, were also discarded.

The striking parallel in the epidemiology, symptomatology and pathology of beriberi and poliomyelitis would seem to indicate some common major etiological factor, suggestively vitamin B deficiency. The fact that practically all of the parallel features may be explained physiologically on the basis of associated increase in vitamin B demand is even more strongly suggestive. It is known, for instance, that the bodily requirement of vitamin B_1 is in direct relation to the metabolic rate. It is known also that the metabolic rate is higher in childhood and early youth than in the adult. It is higher in the male than in the female, higher in the physically active than in the sedentary, and higher in pregnancy than in non-pregnancy. It is known too that the need for this vitamin is much gretater in hot humid weather, owing, perhaps, to its increased loss through perspiration and urine. All these determining factors provide a logical physiological explanation for many of the hitherto inexplicable epidemiological features of poliomyelitis, notably the predominant incidence in childhood, in pregnancy, in hot weather, in the male and in the physically overactive subject.

Since the author first advanced the hypothesis of vitamin B deficiency as the basic cause of poliomyelitis in 1937, clinical trial of vitamin B therapy in this disease has been made[3, 5, 8, 9, 10] in both America and Europe, with results almost as favorable as in the treatment of beriberi by extract of rice polishings. The dietary history has also been investigated by the author in some two hundred cases (Toronto, 1937; Winnipeg, 1941; Los Angeles, 1944), and in every case the dietary intake of vitamin B_1 was found to be definitely deficient. To illustrate the spectacular effect of vitamin B therapy, brief clinical reports of two cases in which it was tried out under the author's direction follow:

Case 1. On October 15, 1938, a fourteen-year-old boy in the Guelph district developed acute poliomyelitis. The onset was with fever, headache, vomiting, sore throat, stiff neck and pain in legs and arms. On October 18 paralysis developed in both legs, back and one arm. On October 20 the patient was brought to Guelph for treatment. Lumbar puncture at this time showed a cell count of 36 and globulin plus. Vitamin B therapy was begun immediately—50 mg. of thiamine hypodermically the first day, 20 mg. on each of the next three days, and 10 mg. on the fifth day. As dietary supplement, marmite (brewer's yeast extract) was given from the start, 12 grams daily. During the first day muscle pains were severe, codiene being required for control. On the third day the pains diminished, and on the fifth day disappeared completely. On the second day the legs were put up in light plaster splints, the arm being left free with instructions not to attempt movement. On November 2 the patient said his legs felt "different." The next day he said his arm felt better, and upon examination he was found to have recovered full muscle function. On November 6 the splints were removed, and it was found that he had recovered full use of his legs also. The following day the patient was discharged.

Case 2. October 18, 1941, a diagnosis of poliomyelitis was made in a six-year-old boy at Niagara Falls, Ontario. The onset was with fever, neck rigidity, muscle tenderness and paresis, and lumbar puncture revealed a definite pleocytosis. Intensive vitamin B therapy was begun October 20—40 mg. hypodermically daily. "Within 24 hours muscle tenderness and rigidity were much improved, and within three days he seemed perfectly normal." There was no residual paralysis or paresis.

The factor of physical overexertion in both beriberi and poliomyelitis is most significant. In the first place, the age incidence of both diseases covers the period of greatest physical activity, the male, generally more active than the female, being predominantly affected. There is also a history of definite overexertion in work or play in most cases of both diseases. Recently Levinson and Sabin[11] have reported that monkeys with experimental poliomyelitis, when subjected to forced exercise or submersion in cool water, or both, developed more severe paralysis than otherwise. De Rudder and Petersen[12] have recently reported a local outbreak of poliomyelitis in a group of school boys, 13 to 19 years of age, shortly following a two-day athletic meet. Five paralytic and eight abortive cases developed among the participants, although nonparticipants from the same school, equally exposed and of more susceptible age, were not affected. Prior and subsequent to this outbreak the town in which these boys lived was free from the disease.

Guerrant and Dutcher[13] have made the interesting observation that growing rats on a vitamin B deficient diet will perform more voluntary exercise in wheel-revolving cages than controls on a vitamin B sufficient diet. When B_1 avitaminosis becomes pronounced there is then a rapid drop in voluntary exercise until the onset of paralysis. This may explain the high-tension overactivity of some children in summer play and the predisposition of such children to poliomyelitis.

Furthermore, in both beriberi and poliomyelitis the muscle groups most in use are usually found to exhibit the greatest degree of paralysis. If the legs are used most, as in marching or bicycle riding, the leg muscles are most prone to paralysis; whereas, if the arms are most in use, as in certain occupations—typists, musicians, painters, etc., the arm and hand muscles are subject to greater paralysis. This may explain why in the human biped the legs are predominantly affected in both these diseases, while in monkeys the arms and tail are mostly affected. Carbohydrate metabolism is directly related to physical activity, and the vitamin B_1 requirement is directly related to carbohydrate metabolism.

A striking example of the selective muscular involvement in poliomyelitis was presented in two cases which the author saw recently in California. One of these, M. L., a boy, aged eleven

years, had been helping his father for several weeks prior to the onset of illness at the work of picking, drying and packing apricots. In this work the arms and hands were mostly used. The residual paralysis and wasting were mostly confined to the corresponding muscle groups. In the other case, R. J. R., a boy of twelve years, gave a history of having done a lot of parallel bar work and rope climbing in his P. T. exercises at school. The objective in the rope climing was to pull the body up, hand over hand, a distance of 15 feet in 9 seconds without using the legs. Needless to say the predominant residual paralysis in this case was also in the arms and hands, with marked griffen-claw deformity in evidence at three months.

Another interesting feature in the selective incidence of both beriberi and poliomyelitis is their marked predilection for pregnant women. Aycock[14] reports the incidence of poliomyelitis to be 450 per cent greater in pregnancy than in non-pregnancy. Top[15] reports that in Detroit the incidence of poliomyelitis was found to be four times greater in pregnant than non-pregnant women of similar age groups. Coupling with these findings the observation of Shute[16] that pregnancy increases the requirement of vitamin B_1 about four times we see the possible etiological correlation of vitamin B deficiency.

A further pronounced affinity between beriberi and poliomyelitis is shown in the predisposing effect of traumata and surgical operations. Bradden[17], reporting on beriberi in the Mediterranean area during the first great war, states: "Beriberi symptoms are not infrequently noted, acute for the most part, after traumata or overexertion. After an operation, severe fatal manifestations occur in persons who, before the operation, showed no symptoms at all." Likewise in poliomyelitis frequent references have been made in the literature to the precipitating influence of surgical operations. In six such cases which the author investigated during the 1937 outbreak in Toronto, three were secondary to tonsillectomy, the other three being secondary to appendectomy, mastoidotomy and tooth extraction. Aycock[18] cites many such cases, mostly tonsillectomy. Francis, Jr.[19] et al. report five severe bulbar cases in the same family shortly following tonsillectomy and tooth extraction, resulting in three deaths. The sole remaining member of the family, a boy of 2½ years, although intimately associated with the

other cases and of more vulnerable age, was exempt. Kessell[20] reports one of the cases in the 1943 California epidemic as having been the immediate sequel of tooth extraction. It is not surprising to find tonsillectomy predominating in this type of case, since it is the operation of major incidence in childhood. It would appear, as expressed in a previous paper,[6] that the precipitating factor in all such postoperative cases, in both beriberi and poliomyelitis, may be the effect of surgical trauma and anesthesia in increasing vitamin B_1 requirement, rather than the generally accepted theory that the denuded site of operation may provide a favorable "portal of entry" for the virus. At any rate this latter theory cannot be applied to postoperative beriberi.

Regarding the patholigical findings in beriberi and poliomyelitis: In both we find marked edema of both the central nervous system and the affected extremities. This condition is often so pronounced that the wasting of muscles is masked until the edematous fluid is absorbed—hence the term "wet beriberi." Some authorities regard the edema of the spinal cord in poliomyelitis as the cause of much of the temporary paralysis, owing to pressure and restricted blood supply. This edematous status in poliomyelitis may account for the frequent observation that victims of the disease are apparently "plump and well nourished." Many of the victims of the Nauru epidemic were reported as exhibiting a "spongy fattiness." In infantile beriberi the edema is such that the body weight increases as death approaches.

Residual paralyses assume almost the same pattern in both diseases, tending to the talipes equinovarus type as a result of paralysis of the anterior tibial and peroneal muscles and spastic action of the opposing muscles. When the upper extremities are involved, which is relatively rare in both diseases, the small muscles of the hand and forearm are generally affected, resulting in the characteristic "griffin-claw" deformity.

Another pathological feature common to both diseases is the marked lymphocytic or mesodermal-glial reaction. In beriberi there is a diffuse lymphocytic infiltration of the perineural tissues generally. In poliomyelitis the lymphocytic reaction takes on a more acute form, the small round cells being found in perivascular clusters—"cuffing." This characteristic distribution of extravasated lymphocytes (noted also in encephalitis and other acute forms of nerve tissue degeneration) may be due to the fact that

these cases usually come to necropsy in the acute stage of a short illness, before the more diffuse infiltration of the lymphocytes is effected as in beriberi; while victims of the latter disease usually come to necropsy in the terminal stage of a prolonged illness. In poliomyelitis there is also a marked lymphatic pleocytosis of the cerebrospinal fluid. A differential cell count of the cerebrospinal fluid in the early phase of the disease usually reveals an increase of the polymorphonuclear neutrophiles, while as the disease advances the lymphocytes predominate. Unfortunately there are insufficient cytological recordings of the spinal fluid in beriberi to be conclusive. The blood findings in beriberi, however, seem to follow the pattern of the spinal fluid in poliomyelitis, as previously described. It would thus appear that the variation in the lymphocytic phenomena in both these diseases might be merely the expression of a different intensity of reaction to the same etiological agent —B_1 avitaminosis.

Howe and Bodian[21] intimate the possibility that the general lymphocytic reaction in poliomyelitis may be a protective response of the organism, and that the elimination of large numbers of lymphocytes in the intestine may be a means of eliminating the virus during convalescence. The fact that other degenerative diseases of the nervous system, including encephalitis, neurosyphilis, spinal sclerosis, beriberi, etc., are accompanied by a somewhat similar lymphatic reaction would seem to indicate that it is related to the degeneration of nerve tissue generally, rather than as a specific response. The recent reporting of lymphatic pleocytosis and increase in protein content of the spinal fluid in a case of oxygen deprivation resulting from carbon monoxide poisoning[22] gives support to this conception, and suggests the possibility that this mesodermal-glial response may in reality be the inception of a reparative reaction to the disintegration of nerve tissue, the differentiation of the extravasated lymphocytes serving, perhaps, to lay the groundwork for the formation of neuroglia for the repair of the breach. On this basis the lymphatic reaction in poliomyelitis may serve as an index of vitamin B_1 deficiency, and may thus be regarded as a reparative response to the destructive action of such deficiency.

Pathological findings similar to poliomyelitis have also been reported in experimental anoxia, as in altitude aeronautics and in

the inhalation of oxygen-displacing gases, such as nitrous oxide, carbon tetrachloride, carbon monoxide, etc. Gerard[23] has shown that the rate of tissue respiration, or oxygen requirement, in the central nervous system is about thirty times that of other body tissues. Van Liere[24] says: "Of all the tissues of the body nervous tissue is the least capable of withstanding oxygen want." It is well-known that there is a lowered oxygen uptake and an increase in the lactic acid content of the tissues generally, and of the central nervous system in particular, in anoxic and vitamin-B_1-deficiency states. Kinnersley and Peters[25] have found that even a small crystal of vitamin B_1 added to an invitro preparation of fresh B_1-avitaminotic brain tissue will quickly increase the oxygen uptake of same. Rosenbohm[26] has found that an accumulation of 30 mg. of lactic acid per 100 Gm. of brain tissue can initiate nerve cell proteolysis. Of further significance is the statement by Van Liere[24] that in severe anoxia there is produced an ascending type of paralysis (similar to that of poliomyelitis). First the legs lose their power, then the arms and neck become affected, followed by bulbar and cerebral involvement. A correlation of these observations may provide a physiological explanation of the sequence of pathological changes common to beriberi, poliomyelitis and other degenerative diseases of the nervous system, and may throw new light on the etiological relationship of postoperative beriberi and poliomyelitis. It may also provide an explanation for the unusually rapid pulse which prevails in both these diseases—a compensatory effort on the part of the heart to overcome the anoxia associated with vitamin B_1 deficiency. It may even explain the mystery of the enlarged heart in beriberi—the effect of the long-continued compensatory effort.

The foregoing study of the comparative pathology of beriberi and poliomyelitis leads the author to the conclusion that the destructive changes in the latter disease are referable to a biochemical disturbance in carbohydrate metabolism, rather than to an inflammatory reaction. Accordingly, the substitution of the term poliomyelopathy is suggested as more correctly expressive of the true nature of the disease.

The metabolic concept of poliomyelitis does not exclude the possibility of the correlated incidence of an infectious process as manifested in the so-called initial or febrile stage of the disease.

This febrile stage may take the form of and may actually be most any acute infectious disease, frequently influenza, the paralytic involvement being regarded as a sequel to the vitamin B_1 depletion brought about by the accelerated metabolic rate associated with the infectious prelude. Holt[27] reports 565 cases of poliomyelitis, in 12 per cent of which the paralysis developed as an immediate sequel to some acute infectious disease of childhood, frequently diarrhea, scarlet fever or measles. The author knows of several cases which developed following pneumonia and one after acute middle ear disease.

A similar situation has prevailed regarding the sequelae of alcoholism (polyneuritis, paralysis, Korsakoff's syndrome and Wernicke's encephalitis). For many years these were thought to be due to the specific effect of alcohol on the nervous system. It is now generally recognized that these neuropathic phenomena are caused by vitamin B_1 deficiency as a secondary effect in alcoholism. Likewise, postdiphtheritic and posttyphoid paralyses were formerly thought to be the direct effect of the specific bacterial toxins; whereas, now they are regarded as due to vitamin B_1 deficiency as a secondary effect of the infectious process.

At the time of the Flexner experiments in transmission of poliomyelitis, Koch's postulates seemed to have been fully met, with the one exception of the culturing of the specific infectious agent; and the most natural conclusion, based on the principles of bacteriology then extant, was that the disease must be infectious and communicable. If, however, the virus be regarded as a biochemical substance associated with the disease process, its origin may indeed be endogenous—the result rather than the cause of the disease, thus eliminating the whole question of communicability, means of spread and portal of entry. Even though this chemical substance, or virus, extracted from the nervous system of a victim of the disease, is capable of reproducing a similar paralytic process in experimental animals upon intraneural inoculation, and possibly by catalytic action as explained by Twort,[28] this, in itself, is not conclusive evidence that the disease is communicable by this means under ordinary circumstances. It is possible that a similar biochemical substance, or virus, may be obtainable from the involved nerve structures in beriberi, and capable, perhaps, of producing similar paralytic symptoms in experimental animals in like manner.

Apparently no research work has yet been done to determine this possibility, although Huffman[29] states "that in recent efforts to locate the virus of beriberi all the organs of the body have been examined and experimented with except the spinal cord, and in each instance the results have been negative." The recent work of Pinkerton and Swank[30] gives support to the author's concept in this respect. They were able to obtain from the nervous system of otherwise healthy B_1-avitaminotic pigeons a substance identical with the virus of psittacosis, capable of reproducing the disease in healthy birds by inoculation. The fact that there has been no case of the spontaneous development of poliomyelitis in monkeys caged with inoculated subjects shows that the virus is not the major etiological factor under ordinary conditions of contact. Likewise in the human subject contraction of the disease by definite contact with another victim of the disease is very difficult to establish, as shown by the elaborate epidemiological study of New York City's 1949 cases, as reported recently by the local Department of Health.

Beriberi has been definitely proved to be caused by deficiency of vitamin B_1, yet as recently as 48 years ago it was regarded as infectious and contagious, in much the same way as the present conventional concept of poliomyelitis. This is shown by the following quotation from Manson[31] (1902): "The proof of the germ theory of the cause of beriberi lies in the circumstance that the disease can be introduced into virgin country and there spread. That is to say the cause can multiply. Spontaneous multiplication is a property peculiar to living things. Therfore, the originating agent of the toxin of beriberi is a living thing—a germ. The toxin of beriberi is not conveyed in drinking water. This is easily proved. Two institutions placed side by side, as for example the male and female prisons in Singapore with identical water supplies; one, the male, is attacked with beriberi; the other, the female, is exempt. We therefore must conclude that the virus of the disease is conveyed to man either by the air or through the skin by contact, or by means of some insect or other animal which inserts it under the skin, or by a combination of these ways. So far, unfortunately, we have nothing to show either what the toxin is or what the germ that produces it is, nor what the precise nidus in which it is produced is, nor the way by which it gains access to the body."

The medical history of pellagra, another disease now known to be caused by nutritional deficiency, has been very similar to that of beriberi. For many years a fungus associated with maize was thought to be the etiological agent. While the disease was thus regarded and accordingly treated the mortality rate was over 50 per cent. Under vitamin therapy and nutritional correction the mortality rate has been reduced almost to zero.

Irrespective of the etiology of poliomyelitis or means of transmission, if any, the associated presence of a biochemical substance which we call a virus, even though it may be capable of reproducing under certain conditions a similar disease in experimental animals, should not be our main concern; but rather the prevention or correction of the preconditioning factors. For, if the virus is as widespread as now generally believed, in the excretions of many hypothetical carriers and consequently in sewage and drinking water, the impossibility of escape from exposure or contacts is obvious. The logical conclusion would seem to be that the great bulk of our population must possess natural immunity to the development of the disease, and the means of acquiring such protection should determine our prophylactic and therapeutic efforts.

Some unusual features of the recent outbreak of poliomyelitis in the Canadian Arctic deserve consideration here. This was one of the most intensive outbreaks, in point of rate of morbidity and mortality, ever recorded in North America. Hrolv[22] reports an outbreak in Greenland in 1914 of slightly greater intensity, resulting in 37 deaths in a population of 700 Eskimos. At that time almost the entire community suffered from an "influenza-like ailment" which in many cases was a prelude to paralytic symptoms. In the recent outbreak, which occurred in the Chesterfield-Inlet district on the northwest coast of Hudson's Bay, Adamson et al.[23] report at least 78 paralytic cases and 14 deaths among a population of 275 Eskimos, within a period of three weeks, February 14 to March 7, 1949. It is roughly estimated that fully 50 per cent of the entire community had symptoms of the disease. In harmony with the conventional theory of communicability, all these cases were traced by devious and dubious routes, through the agency of hypothetical "carriers" and "reservoirs," to one original case of the disease which occurred in Churchill, nearly 400 miles distant, the previous summer; and this in spite of the

relative isolation of the community by rigorous Arctic weather, to say nothing of adverse geographical conditions. The seasonal incidence was unusual as compared to that of our temperate zones, the outbreak occurring in severe winter weather with temperatures ranging from –21° to –49° F. The winter incidence of the disease is not unusual in the Arctic, however, since at this time the hygienic and nutritional status of the Eskimo reaches its lowest level. The age incidence was almost equally distributed in all groups from 5 to 60 years. The under-five-year group was least affected, with only two cases among 53 in this group, and none under three years. From the nutritional viewpoint this seems rather significant, since it is the custom of Eskimo women to nurse their babies until two or three years old, or until another child is born. During pregnancy and lactation Nature mobilizes the nutritional resources of the mother for the benefit of her progeny irrespective of her own needs. (As pointed out in a previous paper[34] it is the author's belief that the gradually lessened incidence of poliomyelitis, and for that matter tuberculosis, diphtheria, scarlet fever, etc., in the infant age group during the last five decades, has been due to the increased use of vitamin-rich food supplements, such as orange juice and wheat-germ-fortified cereals, in pediatric nutrition. The age of greatest poliomyelitis incidence is now in the 5 to 15-year bracket, whereas fifty years ago the most critical age was two years. This disease can no longer be called infantile paralysis.)

Another nutritional feature, which from the viewpoint of the author may have contributed to the unusually high rate of incidence at Chesterfield, is the high proportion of raw sun-dried fish in the Eskimo diet. In 1941 Green et al.[35] reported a widespread outbreak of paralytic encephalitis in fox ranches in Minnesota, which was found to be caused by the feeding of raw fish and to be curable by intensive vitamin B_1 therapy. They concluded that there was some element in uncooked fish that had a neutralizing effect on thiamine.

Another factor which may have contributed to vitamin deficiency among the Eskimos, and thus to the higher incidence of disease, is the fact that in recent years their primitive life has been adversely modified by government measures designed to improve their condition—family allowances, destitute allowances, old-age pensions,

etc. In many of the large Eskimo families these retroactive gratuities brought unexpected relative wealth, so that most families had several hundred dollars with which to purchase the white man's refined foods from the Hudson's Bay Company posts. This resulted in a marked tendency for the natives to huddle around these distributing points during the severe winter weather and live on their new found wealth. In the Chesterfield district during the time of the poliomyelitis outbreak almost 200 of the total population of 275 were found to be living within ten miles of the trading post. The Eskimos' principal food purchases consisted of white flour, white sugar, corn syrup, lard, tea and tobacco, all of which are almost completely devoid of essential minerals and vitamins. In a recent nutritional survey of Northern Manitoba Indians, Moore et al.[36] found that the intake of vitamin C in their usual diets was less than one-seventieth of the daily requirement, besides marked deficiencies in other respects (it is more than likely that the Eskimos, still farther north, are even more deficient), and they concluded that malnutrition might be the major causal factor in the high mortality rate from tuberculosis and other infectious diseases among the Indians. Apparently this conclusion applies equally to the Eskimos.

Another more recent outbreak of "poliomyelitis" (January 1950) has occurred in an isolated community in Labrador in sub-zero weather. First reports indicate 29 cases with 4 deaths in the small coastal village of St. Augustine. Heretofore Labrador has been an endemic center for beriberi. Perhaps disease styles are changing in this locality, or are they?

There is another unusual outbreak of poliomyelitis on record which provides a close parallel with the recent Arctic outbreak. In 1910, on the small south-Pacific island of Nauru, there occurred within two weeks 690 cases with 38 deaths in a native population of 1,750. Dr. Muller, the lone physician on the island, was hesitant in diagnosing the disease as poliomyelitis, since, aside from the neuromuscular symptoms in a portion of the cases, he regarded the epidemic as typical of influenza because of the rapid spread and almost simultaneous incidence. However, some years later many of the victims of this outbreak were carefully examined by a delegation of experts and were found to have residual paralyses and deformities characteristic of poliomyelitis, so that at the present time the identity of the disease is undisputed.

The transmission of the disease to the island could not be established. Vessels from Australia, which had been eleven days in transit, had no cases on board; steamers from Hong Kong, nearly three weeks en route, had no cases, and neither was there any similar disease at ports of call. Natives arriving by these steamers were healthy and remained so through the entire epidemic.

The nutritional and economic background of the natives of Nauru was closely similar to that of the Eskimos. The chief industry of the island was phosphate mining, developed by the Germans, and most of the natives were receiving cash royalties for the mining rights on their properties. This easy money was quickly turned back to the Company stores in the purchase of white flour, white rice, white sugar, canned meats, etc. The women of the island developed the habit of drinking sugar-sweetened water as a pastime, and the opulent men-folk became increasingly lazy and lessened their food sustenance from fish, fresh fruits, nuts, etc. (Such a nutritional program would obviously be conducive to deficiency.) Included in the native population were some five hundred imported laborers from the neighboring Carolines, and these, together with 500 Chinese and 80 Europeans, were all employees of the mining company. These last three groups, having no property rights on the island, were not receiving royalties and were accordingly on a different nutritional program, more in keeping with their traditional native habits. In this connection the incidence and severity of the disease in the different groups was most significant. The 1,250 native Nauruans had 470 cases and 37 deaths, while there were 220 milder cases with only one death among the Carolinese. The Chinese were completely exempt, and there were only three mild cases among the Europeans. The Nauruan children, as in the recent Arctic outbreak, were almost completely exempt, the incidence being confined mostly to age groups from 12 to 40 years. From the author's viewpoint this might be accounted for by the possibility of the children in their play having access to more fresh fruits, nuts and other natural food products of the island.

Following this and other devastating disease outbreaks (tuberculosis, leprosy, influenza and infantile beriberi) on this isolated island a radical change in the native dietary was instituted by the local health officer. The sale of white flour and polished rice was for-

bidden in the native stores, whole-meal flour and brown rice being substituted, and the use of whole-wheat bread was encouraged. Sweet potatoes were introduced and the value of eggs and condensed milk was stressed. The sale of sugar was restricted to one pound per adult per week. Following these measures the infant death rate rapidly fell from 50 per cent to 7 per cent in the first year of life, at which level it has consistently remained. Not only has there been no further reported occurrence of poliomyelitis and infantile beriberi, but even leprosy and tuberculosis, which were rife on the island, have been almost completely eradicated. This is in harmony with the observation of Takaki that whenever beriberi was reduced in the Japanese navy by corrective nutrition the health of the sailors improved generally and the incidence of other diseases decreased. (For a more complete report of the Nauru poliomyelitis episode the reader is referred to the author's first paper on poliomyelitis[2]).

This discussion would be incomplete without reference to the apparently paradoxical findings of a number of research groups on the question of the relation of the thiamine level to the susceptibility of laboratory animals to experimental poliomyelitis. In 1944, Foster et al.[37] reported an apparently increased resistance to the Lansing strain of poliomyelitis virus in experimental mice on a vitamin-B_1-deficient or restricted food intake, but admitted that their findings were difficult of interpretation and inconclusive and intimated additional experimentation was needed to clarify the problem. Later in the same year, Toomey et al.[38] reported a series of parallel experiments on laboratory animals in which the findings were apparently reversed. They concluded that "at first glance it would seem that fewer of the under-fed animals developed the disease. This need not be true, however, since certainly there was no evidence that the deficiency spared these animals. . . . Actually a conclusion reverse to the usual might be made and that it might be suggested that B_1-over-fed mice developed some slight protection." In 1945, Clark et al.,[39] reporting on the influence of thiamine deficiency on susceptibility of monkeys to experimental poliomyelitis, as studied in forty animals, conclude "that this species, when deficient in thiamin, does not exhibit an increased resistance to poliomyelitis virus." In 1946, Weaver,[40] reporting on the "resistance of cotton rats to poliomyelitis virus as influenced by intake of vitamins" pointed out that "the difference in response

shown on the one hand by mice and on the other hand by cotton rats, even though they were subjected to similar nutritional stresses and inoculated with the same strain of the virus, illustrate the danger of applying directly to human beings the results of animal experimentation."

Since, therefore, the anthropology of the human more closely parallels that of the monkey than that of rats and mice, the above series of experiments fail to invalidate the author's hypothesis.

A much more significant finding regarding the effect of restricted food intake on poliomyelitis incidence was made on a much larger scale and on human subjects of the 1937 Toronto outbreak. At that time there were about 50,000 of the population on the relief diet supplied gratuitously by the city. (Sometime later a committee of nutritionists investigating this diet reported it grossly deficient.) The remaining 600,000 of the population, who might be regarded as controls, were not on municipal relief and obviously had a more liberal diet and higher intake of thiamine. There were 184 cases of the disease among the reliefees and 562 cases among non-reliefees. The ratio of incidence was four times higher among the reliefees on the restricted food intake.

In conclusion, we quote from a recent letter to the editor of the Journal of the American Medical Association, as follows: "Sabin asks in his article on the Epidemiology of Poliomyelitis (J. A. M. A., June 28, 1947) why did paralytic poliomyelitis become an epidemic disease only a little more than 50 years ago, and as such why does it seem to affect mostly the countries in which sanitation and hygiene, along with general standards of living, are presumably making the greatest advance, while other large parts of the world, regardless of latitude, are still relatively unaffected? My answer: Let us take the hypothesis that B-hypovitaminosis is a disposing factor for poliomyelitis paralysis. The general B-hypovitaminosis induced by milling fine flour and other changes in nutritional habits is followed in time by the appearance of the first poliomyelitis epidemic, apart from the fact that the countries with the best developed hygiene are those with pronounced B-hypovitaminosis. I am sure this is only one part of the problem. In any case it would be worth while to carry through on a large scale with your incomparable possibilities in schools, etc., by additional vitamin-B supply." Signed: C. Braendli-Wyss, M.D., Burgdorf, Switzerland.[41]

The author agrees with Braendli-Wyss that the best way to solve this problem would be to make a mass trial of the prophylactic effect of an optimal intake of vitamin B_1 in the human susceptible age group. Such an experiment would certainly be fraught with less danger than was that of the mass trial of the zinc-sulphate nasal sprays in the Toronto outbreak of 1937. In any event it could have nothing but good effect, since vitamin B_1 deficiency is the recognized major defect in our modern diet.

SUMMARY

The marked resemblance in the epidemiology, symptomatology and pathology of poliomyelitis and beriberi is presented in support of the author's hypothesis of vitamin B_1 deficiency as the basic etiological factor in poliomyelitis. As additional evidence a close parallel is drawn between the recent poliomyelitis outbreak in the Canadian Arctic and that of the island of Nauru (1910) from the nutritional angle.

The modern concept of the virus as a chemical substance, a protein molecule of definite dimension, rather than an ultramicroscopic organism, as previously thought to be, calls for a revision of the interpretation of the Flexner experiments as to specificity and transmissibility of the disease, with consideration of the possibility of the endogenous or metabolic origin of the virus.

It is suggested that the paralytic phenomena in poliomyelitis may be indirectly referable to acute B_1 avitaminosis precipitated by converging factors which increase the metabolic rate and accordingly the bodily requirement for this vitamin, such as febrile conditions, age, sex, climatic conditions, physical overexertion in work or play, and pregnancy; and that the paralysis may be a secondary metabolic manifestation rather than a primary infectious disease.

The associated virus is regarded as a biochemical substance produced by the disease, which, although capable of producing paralysis in experimental animals by catalytic chemical action in the nervous system, is not necessarily a means of spread of the disease under ordinary conditions. It is suggested that a virus-like substance may be obtainable from the nerve tissues of beriberi victims—an experiment which apparently has not yet been attempted.

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PSEUDOHERMAPHRODITISM IN TWINS*

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In 1943 Rhodes[1] reviewed the literature on pseudohermaphroditism in twins, reporting on 9 cases and adding a new case occurring in identical twins. The case of Collier's[2], in 1948, makes a total of 10 cases in the literature. This paper is the follow-up of the case reported by Rhodes.

At time of birth, the twins described by Rhodes were considered to be female. However, upon examination seven weeks after birth, it was Rhodes' impression that these children were male pseudohermaphrodites. This was based on careful examination of the external genitalia (Fig. 1.). It was recommended that further investigation be carried out at a later date to definitely determine their sex. Collier also states that early diagnosis and plastic work is desirable and surgical therapy should be performed at a preschool age.

Following this recommendation, one of the twins (L. T.) was readmitted to Flower Fifth Avenue Hospital in April 1946 for exploratory laparotomy. The abdomen was opened through a small Pfannenstiel incision and a normal uterus, tubes and ovaries were found. There was no evidence of male gonads. Biopsy taken from the left ovary was reported as histologically normal. Careful examination, while the child was under anesthesia, revealed the presence of and patency of a vagina just under the urethral meatus. The clitoris measured 2 cm. in length.

The sex of the children having been definitely determined, it was decided to follow them carefully and do a plastic repair of the external genitalia if this should prove necessary at a later date.

In September 1948, follow-up examination on the now six-year-old twins revealed normal vulvae, urethrae and hymens. The clitori were hyperthrophied measuring over 4 cm. in length (Fig. 2). The children were healthy and normal in all other respects. On the basis of gynecolgic, urologic and psychiatric

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consultations it was decided that plastic repair of the hypertrophied clitori was indicated at this time, particularly since the twins were due to start school.

The children were admitted to the hospital on October 18, 1948, for surgery, and the operations were performed in the same

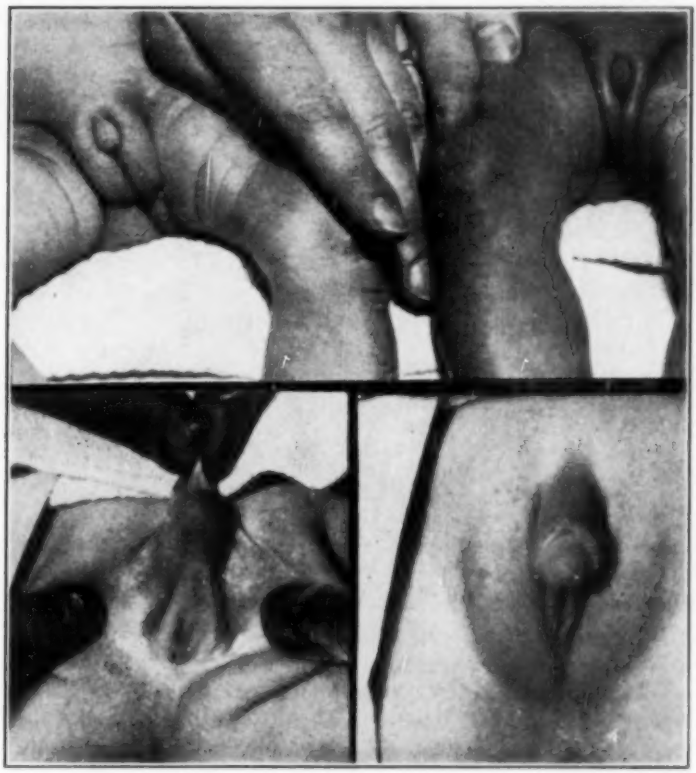

Fig. 1. (top) Appearance of external genitalia at seven weeks of age.
Fig. 2. (bottom) Appearance of external genitalia at six years of age. (a) Vagina and urethra below clitoris. (b) Hypertrophied clitoris.

manner for both. The clitoris was pulled downward and an eliptical incision was made from the base down on each side along the prepuce as far as the labia minora. The prepuce was

then dissected back by sharp dissection. The underportion of the clitoris was freed as far back as possible and the clitoris was then amputated near its base. The cut edges of the fascia of the clitoris were approximated with over and under sutures. The upper part of the incision was approximated so as to form a superior commissure covering the amputated clitoris. The remaining prepuce was sutured to the cut edge of the labia minora. The resulting appearance was that of an intact labia majora and an intact labia minora with the clitoris and most of the prepuce obliterated (Fig. 3).

Fig. 3. Appearance of external genitalia after plastic repair.

The children made uneventful recoveries and are still being seen in the follow-up clinic.

The amputated clitori were sent for microscopic examination which revealed well-formed cavernous sinuses and good dorsal vessels and central arteries. No corpus spongiosum was noted.

SUMMARY

A follow-up of a previously reported case of pseudohermaphroditism in twins was presented. Definite determination of the sex was made by exploratory laparotomy on one of the twins. On the basis of this and subsequent findings, a plastic amputation of the hypertrophied clitori was performed. It is felt that the opera-

tion was performed at the optimum time for future normal adjustments. The technique of the operation has been described.

The authors wish to take this opportunity to thank Dr. Leon Loizeaux, Director of Obstetrics, and Dr. Sprague Carleton, Director, Post-Graduate Urology, for their help and guidance in the management of this case.

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SOY BEAN MILK IN FEEDING OF INFANTS. (Indian Journal of Medical Research, Calcutta, *37*: *77*, Jan. 1949). Desikachar and Subrahmanyan found that rats and adult human subjects easily digested and absorbed milk and curd prepared from soy beans. Children under 1 year were given soy milk as the sole food, while older children received it as a supplement. All the children took readily to soy milk. The growth rate was normal, and no digestive disorders could be observed. Absorption and utilization of the proteins, calcium and phosphorus in cow's milk and soy milk were compared, and it was found that soy milk protein was 86 per cent as utilizable as cow's milk protein. There was practically no difference in the utilization of calcium and phosphorus in the two kinds of milk.—*Journal A.M.A.*

COMPLICATED VISCERAL ABNORMALITIES. (Archivs Argentinos de Pediatria, Buenos Aires, 20: 263, June, 1949). The subject of this report is a 21-month-old girl with cyanosis from birth. There was neither tuberculosis nor syphilis in the history of the family. The mother and older brother of the patient had congenital aniridia. At the age of 21 months the patient suddenly presented left hemiparesis. Examination of the patient revealed transposition of the viscera, levocardia, cardiopathy and cystic disease of the lung. Pneumonectomy was performed on the assumption that hemiparesis was caused by an embolus from the cystic lung. This was followed by improvement in the blood, and almost complete disappearance of cyanosis and of the cardiorespiratory difficulties. The patient was well as of the time of reporting, one year and three months after the operation. The auricles were not transposed, notwithstanding the fact that the inferior vena cava followed an abnormal course on the left side of the stomach.—*Journal A.M.A.*

ATYPICAL PYLORIC STENOSIS*

Report of Two Cases in Negro Infants in Whom the Vomiting Began on the First Day of Life

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In the usual case of congenital hypertrophic pyloric stenosis, symptoms are not apparent until 2 to 6 weeks after birth. The condition is, therefore, not often considered in the differential diagnosis of vomiting occurring in the first few days of life.

Wallgren,[12] who did barium studies on 1,000 normal newborn males, found no evidence of the disease in any of the infants including five who later developed typical pyloric stenosis.

In reviewing the literature for the past twenty years, we have found reports of 30 cases in which diagnosis was verified at operation and in which the vomiting was definitely stated to have begun at birth.

The general incidence of the condition is stated to be 2 to 4 per 1,000 live births. Observers agree that it is less common in the female (10 to 20 per cent of cases) and in the Negro (1 to 2 per cent of cases). For the year 1943, Children's Hospital of Washington, D. C., had an incidence of pyloric stenosis of 0.14 per cent of Negro admissions (3 cases in 2,159 admissions) and 0.65 per cent of white admissions (27 cases in 4,319 admissions). The incidence at Freedmen's Hospital for the five-year period, 1943-48, was 0.14 per cent of pediatric admissions, all Negro (4 cases in 2,917 admissions). Ladd[8] observed no racial predisposition to the condition in Boston.

We wish to report two cases, the unusual features of which are that both are Negroes and in both the vomiting began with the first feeding after birth.

CASE REPORTS

Case 1. S. B., female, born November 21, 1947, and admitted to the pediatric ward at the age of 8 days with a complaint of vomiting feedings since birth.

The infant was delivered at home in rural Maryland by a mid-

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wife. Birth weight was said to be 9 pounds. An evaporated milk formula was given. Vomiting occurred with the first feeding. All subsequent feedings were followed by projectile vomiting. Vomitus contained no blood or bile. Two to three small yellow stools were passed daily.

On admission the temperature was 98° F. and the weight 5 pounds 15 ounces. Emaciation and dehydration were marked. The abdomen was distended and tympanitic. Peristaltic waves could be seen passing from left to right across the epigastrium. A hard acorn-size mass was palpated deep in the right upper quadrant.

On admission the blood nonprotein nitrogen was 54 mg. per cent and the total protein 6.27 per cent. Hemoconcentration was present but after hydration the blood count was normal. Urine and spinal fluid were normal. The blood Kahn was negative.

Supportive therapy, consisting of stimulants, subcutaneous and intravenous fluids, followed by plasma and blood transfusion, caused a marked improvement in the general condition. Feedings, consisting of a dryco formula were eagerly taken but not retained. Phenobarbital and eumydrin had no effect on the vomiting. Stools were green and scanty. A 13-ounce weight gain followed the restoration of fluid balance. Thereafter the weight remained stationary.

X-ray showed a greatly dilated stomach. No barium had left the stomach after three and one-half hours.

Laparotomy on December 9 revealed a typical hypertrophy of the pyloric muscle. A Fredet-Ramstedt pyloromyotomy was performed.

Feedings were begun 24 hours after operation. Vomiting began again and persisted. Vomitus contained bile. A flat plate of the abdomen showed distended loops of small bowel suggesting intestinal obstruction. The abdomen was again explored on December 12 and there was found a small bowel obstruction due to volvulus and adhesive bands. The gut was viable and the obstruction was relieved. Postoperatively the infant received intravenous fluids and small amounts of saline by stomach tube.

Eight hours after operation she vomited, became cyanotic and dyspneic, and died a few hours later. Death was presumably due to aspiration of vomitus. Post-mortem x-ray of the chest was negative. No autopsy was performed.

Case 2. M. M., a full term male infant, was born at Freedmen's Hospital on November 7, 1947, weighing 6 pounds 13 ounces. The infant appeared normal at birth.

Twelve hours after birth an evaporated milk formula was given and immediately vomited. He was changed to a low fat and then to a cereal thickened formula, but vomiting persisted. Atropine and phenobarbital were ineffective. Normal meconium stools were passed. No abdominal tumor was palpated. The child was maintained in fairly good condition by means of parenteral fluids and blood transfusion.

Blood counts and urinalysis were normal. X-ray of the abdomen on November 11 showed a large amount of gas in the stomach and small intestine. A diagnosis of partial intestinal obstruction was considered.

On November 12, when his weight had fallen to 6 pounds 6 ounces, a laparotomy was performed. A pyloric hypertrophy the size of a peanut was found. Pyloromyotomy was performed. The postoperative course was uneventful.

COMMENT AND SUMMARY

We have presented two cases of congenital hypertrophic pyloric stenosis in newborn Negro infants. As has been observed previously, the condition is apparently rare in the Negro.

Pyloric stenosis deserves consideration among the causes of vomiting in the first few days of life. In these atypical cases, where vomiting and regurgitation begin during the first day or so after birth, one, of course, has to exclude other congenital obstructive conditions of the alimentary canal, such as esophageal, duodenal or intestinal anomalies. Intracranial birth injury also has to be considered in the differential diagnosis.

In typical cases of pyloric stenosis, roentgenologic examination is not necessary to establish the diagnosis; however, this method of study may supply valuable corroborative evidence in atypical cases. Should the physician elect to employ contrast medium (barium) in the roentgenologic examination of newly born infants exhibiting vomiting, certain precautions should be observed in reference to the infant's safety and the interpretation of results. These include: (1) Very thin barium mixtures should be used to avoid complete plugging or occlusion of stenosed areas in the

alimentary canal. (2) Precautions should be taken to minimize the chances of aspiration of barium contained in the vomitus. (3) In evaluation of the "emptying time" of the infantile stomach, close observation and scrutiny are necessary to detect spurious partial or complete "emptying" by peroral vomiting. Moreover, according to Caffey,[2] the gastric emptying time may normally be as long as eight hours during the first two weeks of life.

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STREPTOMYCIN IN TUBERCULOUS MENINGITIS. (Maandschrift voor Kindergeneeskunde, Leyden, 17:77, 1949). Van Creveld and Stoppelman say that, from March 1948 to May 1948, 7 children with tuberculous meningitis were treated at the Children's Clinic of the University of Amsterdam. The ages of the children varied between 15 months and 6 years. The children were treated with streptomycin in daily doses of 45 mg. per kilogram of body weight, divided into six intramuscular injections. Three children were also given intraspinal injections of 100 mg. of streptomycin. Four of the 7 children died. Of the 3 surviving patients, 2 children, aged 19 months and 6 years, show severe mental defects. The only child who is now in good health was 15 months of age at the time of onset of the disease.—*Journal A.M.A.*

THE RELATIONSHIP OF TRAUMA TO SICKLE CELL ANEMIA*

Report of Case

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A case of proven sickle cell anemia with a history of trauma and recovery is reported.

CASE REPORT

M. G., colored male, age 7 years, was admitted to City Hospital on April 8, 1949 and was discharged as improved on April 28, 1949. Patient fell in street, three days prior to admission. He walked home, was confined to bed and has refused to move about since. For the past thirty-six hours he complained of a great deal of pain over the entire abdomen. He had no vomiting and had a bowel movement the morning of day of hospital admission.

Examination. Lungs clear; systolic apical murmur. Child held himself rigidly. There was an exaggerated lumbar lordosis and exquisite abdominal pain with nuchal rigidity and opisthotonus. Babinski negative; reflexes plus two throughout on both right and left sides.

Laboratory. Colloidal gold: 0011432100. Quantitative Kolmar: 0.5 neg., 0.25 neg., 0.125 neg., 0.06 neg., 0.03 neg. Type O, comp. 02; Rh positive; Mazzini negative; Urea 9.4; icteric index 22. Blood morphology: R.B.C.3,200,000; W.B.C.25,200; lymphocytes 20; hemoglobin 8 gms. April 8, 1949—Sickle cell preparation positive for sickling. April 11, 1949—Sickle cell preparation positive for sickling in 15 minutes.

Diagnosis. Sickle cell anemia.

Treatment. Transfusions: 150 cc., 250 cc., 250 cc.

DISCUSSION

Sickle cell anemia is an hereditary and familial form of chronic, hemolytic anemia essentially peculiar to Negroes. The sickling of red corpuscles has been observed almost exclusively in the Negro race or in individuals whose blood contains an admixture of Negro blood. Prostration and abdominal tension may be associated, and the picture may so closely simulate that of one of the

*Service of Dr. Isidor Kross, City Hospital, Welfare Island, New York.

abdominal emergencies that many patients have been operated on with the expectation of finding a ruptured peptic ulcer, intestinal obstruction, or some other serious accident which requires surgical intervention. The leukocytosis which is usually found in patients with this disease contributes to the confusion. In one of our cases the initial complaints suggested poliomyelitis. The symptoms may suggest abdominal disease, rheumatic fever, osteomyelitis or neurological disorder. Sickle cell anemia is a serious malady with an ultimately fatal outcome although some patients live on for many years in spite of severe anemia. Most patients die in the first decade of life and very few survive the third. It has been pointed out that death may occur following one of the adbominal crises, this being attributed to shock produced by the rapid removal of red cells from the circulation subsequent to their sickling. The prognosis following central nervous system injury is poor, but it varies and is unpredictable.[1] Itano and Paulding[2] state that six to ten per cent of Negroes possess the sickle trait; their red blood cells have a capacity to sickle, but most of these individuals do not develop anemia. Sickle cell anemia is a congenital chronic hemolytic type of anemia characterized by the development of oat-shaped and sickle-shaped erythrocytes. The moist stasis method, in which blood is obtained from a patient's finger after its circulation has been occluded for five minutes, gives the most rapid and consistent results, and is described.

The symptomatology suggested acute abdominal pathology, meningitis, osteomyelitis of the spine or the prodromal stage of infantile paralysis. X-ray studies of the skull and spine were negative. Observation and laboratory tests made the diagnosis clear.

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PEDIATRICS HALF A CENTURY AGO

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

ACUTE MYOCARDIAL INSUFFICIENCY IN SOME INFECTIONS IN CHILDREN*

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The term, chronic myocardial insufficiency, was introduced by Ebstein, as applying to all those conditions in which the normal relation between pulmonic and systemic circulation is disturbed on account of some anatomical change in the myocardium. In acute myocardial insufficiency the same definition holds good, except in that the process is an acute one. The importance of our subject lies in the fact that a large number of these acute myocardial insufficiencies develop in connection with the infections in childhood—scarlatina, diphtheria, rheumatic fever, septicemia, typhoid fever and pneumonia. Beyond the possibility of a fatal termination from the acute myocardial condition, there remains another which is very far-reaching for the future of the individual who survives. In the greatest number of cases of heart weakness developing during pubescence and young adult life, the only explanation that has presented itself is heart strain, either due to development or inherent, i.e., congenital, myocardial weakness. In some of the diseases mentioned before, especially diphtheria, scarlatina, typhoid fever, the myocardial changes, especially the interstitial ones, are so well marked that permanent danger is done to the heart which may only manifest itself when an extraordinary call upon the functional activity of the heart is made. The view which is daily gaining more ground, that so-called idiopathic dilatations of the heart are not so common as was supposed, therefore, is probably true, the basis for a dilatation being some myocardial change. As it is a fact accepted on all hands, that, in every case of peri- or endocarditis, the myocardium is more or less in-

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volved, it will be seen how great the number of cases becomes in which it is possible that acute myocardial insufficiency may develop. In pericarditis with effusion the additional element may be the possible failure in diastole; in endocarditis, the chance that a damaged myocardium may not be able to perform compensation for a valvular lesion. Furthermore, in acute nephritis of scarlatina, the increased resistance in the left heart will be less readily overcome by a damaged than a healthy myocardium. But the development of symptoms of acute myocardial insufficiency depends upon the amount of damage done to the myocardium, so that in a number of cases no symptoms develop at all; in a much larger number the symptoms develop after the febrile course of the primary disease has ceased, being due to a continuance of the process and its bad results; in these cases the development of symptoms may be postponed indefinitely; in a certain number of cases the changes in the myocardium are so great that they manifest themselves early during the febrile period.

Of the frequency of myocardial changes in connection with infectious diseases there is no doubt since the adoption of Krehl's method for examining the heart. E. Romberg has found myocarditis present in 6 out of 11 cases in typhoid fever (ten children; in 7 out of 88 cases in scarlatina (six children); 2 cases of scarlatina nephritis and in 4 cases out of eight in diphtheria (children); in 4 cases examined by Ribbert, myocardial changes were found; in the latter disease myocardial changes are referred to by a host of authors, French, German, English and American, beginning with Bristowe, in 1859, and ending with Heubner, in 1903. Arthur Neumann (1900) examined seventy children for dilatative heart weakness amongst which number he found eight affected. I was interested in reading the short histories appended to the article to find that six had had infectious diseases; these had had diphtheria alone or diphtheria and other diseases, one scarlatina alone; one had tuberculosis, which need not be considered, and in one only there was a negative history of some infection.

The mechanism of the production of acute myocardial insufficiency is very simple, then; some damage done the myocardium; immaterial whether primarily an interstitial myocarditis (E. Romberg) or primarily a degenerative process and secondarily an interstitial process (Ribbert). Nor does it interest us for the pres-

ent to discuss whether it be the toxins or the bacteria which produce the troubles (Lees). When a sufficient change takes place in the myocardium so that it becomes insufficient, then the reaction on the part of the heart as a whole is sufficiently well marked to assume a distinct clinical picture. At first there is noticed a diminution in heart force and arryhthmia; then follows dilatation of the left heart, then of the right heart; with relative insufficiencies of their valves; as soon as the right heart is seriously affected, marked enlargement of the liver. I have seen the latter descend to the level of the umbilicus. Sometimes the right heart is affected first; but not commonly, as affections of the myocardium follow the rule of all acquired, noncongenital diseases of the heart, viz., that they are more common on the left side. If to a damaged myocardium there be added a predisposing cause, especially affecting the right heart, as whooping-cough or pseudopertussis, the right heart first becomes insufficient. The signs and symptoms vary in different diseases, as will be shown further on, but upon the whole there is produced weakness, irregularity of pulse and heart impulse—tachycardia or bradycardia; a broadening of the heart dullness and bruits heard most commonly over the apex, the mitral and the tricuspid area, but frequently, also, over the base. To this class belong the 2 cases I referred to in my contribution to Jacobi's Festschrift; in 1 case the myocardial change was obesity; in the other it was due to three attacks of influenza, for which disease Huchard has established myocardial changes.

Diphtheria, of all the acute infections, most frequently produces myocardial insufficiency. Schmaltz claims that acute myocarditis occurs in from 10 to 20 per cent of all cases of diphtheria; fortunately, not all of these lead to myocardial insufficiency. The process of myocardial insufficiency, which has been confounded with other methods of heart death in diphtheria, does not begin before the end of the first week of the disease, is most common during the second and third week and may develop as late as the sixth or eighth week. The symptoms are those already described, i.e., when there is only diphtheritic myocarditis. The patient dies when the diseased myocardium can no longer overcome the demands made upon it, especially from distention; primarily then from heart paralysis; but in addition, secondary effects upon in-

nervation and possibly, upon oxygenation, although the latter is rare, as is evinced by the absence of cyanosis. But not enough stress, in my opinion, can be laid upon the fact that pure myocarditis is not the only cause that produces heart death in diphtheria; there are at least two other processes that complicate it. The first of these is the effect of the diphtheria toxin upon the cerebral vasomotor centre first discovered by a series of ingeniously conducted experiments by Romberg and Pässler and fully verified by Heinike, Rolly, v. Stejskal. As the result of this action, paralysis of the vasomotors takes place; therefore, dilatation of the blood vessels, with the result that the blood is not propelled normally. Unfortunately, the parts supplied by the splanchnic nerves will contain so much blood, the liver alone containing one-quarter of the whole amount of the blood in the body, that circulation becomes an impossibility. The result is, that the heart simply "bleeds itself" into the splanchnic area, and when, after a time, very little or no blood flows into the right heart, all of it being stored up in the vessels of the abdominal cavity, systolic contraction ceases. Again, here the symptoms are fairly characteristic. In diphtheria it is the form of heart death that occurs very early in the disease, as I have seen it as early as the third day; this before the days of antitoxin. The dominant symptom is increasing rapidity of the pulse, not marked changes in rhythm, but continuous diminution in filling of the arteries, so that the pulse becomes weaker and weaker, the arteries more and more empty, until it no longer can be felt. The condition of the heart is negative; no dilatation, no bruits, no stases anywhere, therefore no enlargement of the liver. But, again, here a continued diminution in force and a constant increase in rate; this is characterized by dullness of heart sounds, especially the first; and, finally, by a cessation of the heart's action. The aspect of the patient is more or less characteristic; pale, more or less restless, no brain symptoms, i.e., no unconsciousness. The patient feels weak and is unwilling to move. This form of heart death, in pneumonia, I have seen in infants; the abdomen becomes much enlarged. Romberg and Pässler have shown that the diplococcus pneumoniæ produce the same results upon the vasomotors as the diphtheria bacillus.

The whole process, when thoroughly developed, runs its course in a short time, from 6 to 18 or 24 hours.

The second method, complicating the clinical picture, is an affection of the vagus. At the present day the myocardial origin of symptoms, following the physiological research work of Gaskell and of Engelmann, is so much in the ascendant, that the origin of symptoms, because of affections of the vagus, is rather frowned upon. The most recent physiological investigation of Pawlow, in that he kept dogs alive for over five months after bilateral vagotomy without producing changes in the heart, seems to confirm this view. But that the vagus does have an effect upon the human heart is shown by the experiment of Czermack upon himself. But even Engelmann shows that nervous influences play a decided rôle in influencing the number of cardiac contractions, and Lommel, in a most exhaustive paper on arrhythmia, has come to the conclusion that "organic heart diseases never produce it"; they are simply neuroses and, as he has seen it, may be produced by irritation of the vagus centre in diseases of the brain. The latter point is especially valuable, as he says, in permitting us to differentiate organic diseases of the heart from functional ones. It is thoroughly established that, in diphtheria, the vagus is frequently affected, and it is also thoroughly accepted that in a large number of infectious diseases we have arrhythmia. If these two facts be put together with the new ones established by Lommel, we can certainly not be far wrong in stating that in diphtheria arrhythmia may be due to disturbance of the vagus tonus. The next question that arises is the possibility of producing heart death by vagus affection in the human being. It must be confessed that we have no evidences upon this subject, for Czermack, after having caused his heart to stand still by pressure upon his vagus, always come to again. But the little that has just been said will certainly justify the conclusion that, even if the vagus may not produce heart death, the effects of its irritation in a myocardially insufficient heart may hasten the fatal outcome. For therapeutic purposes the fact that myocardial insufficiency in diphtheria may not be a pure process, but complicated, at least, by the two other factors just discussed, seems to me to be very important.

Next to diphtheria my own experience with acute myocardial insufficiency has been most extensive with the septicemia of influenza. Here again we have a complex process, the result of infection with the influenza bacillus and secondary infections with pus producers, notably streptococcus and the various staphylococci; in by far the greatest number of cases the secondary infection coming from the respiratory tract, but sometimes from the gastrointestinal tract. I have seen no case in connection with influenza in which the mechanism of Romberg and Pässler seemed to play a very important rôle, although the influenza toxin seems to have a direct effect upon the vasomotor, as is shown by the change in the pulse temperature ratio. All the cases I have seen have been accompanied by more or less cardiac change. The worst case in children that has come under my observation which has recovered I herewith report.

A. A., seen in private practice, aet. seven years. Family history, tuberculosis on mother's side, neuroses on father's side. The child was bottle-fed as an infant with normal results; she has had one previous attack of mild influenza; otherwise she has been well. On October 27, 1902, I was first called to see her. There was present a house epidemic of influenza, she being the last one of the children to come down with it. It was the ordinary respiratory form, but in a short time developed into pseudopertussis, for which she received quinia hydrobromatum with good results as far as the number of attacks was concerned. On the morning of November 7 I was again called to see her. The statement was made that she had passed a restless night and had seemed very feverish. Upon examination there was a found a temperature of 105.4°F., pulse 122 and respiration 34. No evidences of any organic lesion, even the cough had disappeared. Notwithstanding hydrotherapy, the temperature reached 106.5°F. that evening, with a pulse rate of 140 and respiration 40. The patient became unconscious, took medicine with difficulty, became jerky and was very nervous. At eleven in the evening she became partially conscious, the temperature having been reduced to 104°F., pulse 120, respiration 32.

November 8. May be summed up as beginning with a temperature 102.6°F. in the morning, the pulse remaining at 140, and the respiration 32. The maximum of temperature that day

was 105.4° F., pulse and temperature not varying much from the morning's rate. The nervous condition improved, as the temperature came down the cough began to reappear, all other functions being normal. The urine contained a small quantity of albumin, nothing being found microscopically. Throughout the course of the disease there was no evidence of nephritis. On my evening visit I found the heart dullness somewhat increased, no bruits. At noon massive doses of unguentum Credé were used. The night was better than the former one, but still some delirium upon awakening.

November 9. Found the patient with a morning temperature of 99.2° F., pulse 98-105, respiration 24. The maximum temperature was 100.2° F., pulse 100-104, respiration 24-26. Cough has reappeared, nervous symptoms gone, but the pulse was decidedly irregular. For this reason stimulants were given. Increase in heart dullness seemed more marked than before.

November 10. The temperature in the morning was subnormal, pulse from 84-88 and intermittent, respiration 24. From this time on for nearly two months we had subnormal temperature in the morning. The pulse even on this day sank to 72 and rose to as high as 96. The heart as before.

November 12. I found the child pale, cyanosed, breathing with difficulty and sweating profusely. Pulse was irregular, from 74-78, and heart exertion would bring it up to 100, the pulse becoming emptier and more irregular. Examination of the heart revealed heart dullness beyond the right border of the sternum, left auricle enlarged, systolic bruit at the apex, and a diastolic bruit at the base. Liver enlarged so that its free border could be felt 2 cm. below the free border of the ribs and painful upon pressure. By means of absolute quiet, stimulants, codein for the cough, strophanthus, then resistant movements, the child was able to sit up in bed on November 21 for the first time. During this time the pulse varied from 64 to 88, the former representing a bradycardia in a child. The peculiarity of the pulse, as far as rate is concerned, is best represented by the record on November 21, when she was allowed to sit up in bed. Under observation for an hour, the pulse rate, counted every fifteen minutes, was 72, 76, 78, 78. When she sat up in bed the second time, on the same day, the pulse varied from 88 to 100, showing

the peculiar irritability of the heart's muscle. The hepatic sign of myocardial insufficiency lasted only eighteen hours. So unreliable was the heart's action, although the dullness had diminished and the bruit at the base had disappeared, that I did not venture to allow the child to get up until December 6, 1902, about one month after the onset of symptoms; permitting her then to sit up in bed before allowing her to get up, for three hours at a time, her pulse being watched at regular intervals. In the beginning of her period of sitting up in bed, if, as was natural in a child of her age, notwithstanding all precautions that had been taken, she would move rapidly, her face become pale, she would again sweat freely and the heart dullness would again increase. On the day before she got up, being allowed to sit up in bed for three hours, the pulse was 98 at the beginning and 80 at the end of the time. It is interesting to note here, that for some time any exertion would manifest itself by a reduction in pulse rate; this lasted for eight days, and it was only after this manifestation had disappeared, that I felt safe in pushing the heart to further exertion. On December 23 she was allowed to walk out of her room for the first time, and by this time the bruit at the apex was very faint, no enlargement of the heart could be detected. On Christmas day she was allowed to walk downstairs, her pulse now varying from 90 to 94, but still intermittent; in five days after this she was allowed to take a drive; January 9, 1904, she was allowed to walk upstairs and was then discharged. I took occasion to examine her occasionally and found her pulse still continuing to intermit, the bruit gradually disappearing and the heart losing its irritability. I examined her for the last time on May 17, 1904. She goes to school, romps about with the other children, does not have dyspnea on exertion and seems to be perfectly well. Her heart is of the normal size, there is no bruit anywhere and none can be brought out by exertion. The mother tells me that her pulse still intermits occasionally, but I could not find it so after having examined it for five minutes.

According to my view, we here had a myocarditis, producing acute myocardial insufficiency as the result of a septic infection engrafted upon an influenza. The bruits were due to relative or muscular insufficiencies, the great irritability of the heart especially speaking for myocardial trouble. It seems to me that

this can be looked upon as a pure case of myocarditis, although the possibility of an endocarditis cannot be absolutely excluded. Contrary to Romberg's view, I believe that all the symptoms can be explained upon the basis of this assumption; even the bradycardia which we know to be due, at times to fluctuations of intracardiac pressure, diminution (Ludwig and Thiry) or great increase (Heidenhain) may be explained on this assumption. The absence of edema, one of the points that Romberg insists upon as militating against the existence of myocarditis in influenza, is explained by the fact that the myocardial insufficiency is not persistent. The pallor and sweating is due, not to primary vasomotor disturbances, but to sudden changes in the centres, the result of the dilatation of the heart. But the fact that other processes interfering with circulation exist in influenza cannot be denied.

It would extend this paper beyond the proper limits to discuss acute myocardial insufficiency in connection with scarlatina, typhoid fever or rheumatic fever; in all these affections myocarditis is not uncommon, acute myocardial insufficiency, however, is rare in children; although I have seen one fatal case in scarlatina with scarlatinal nephritis.

Our treatment has been added to since Romberg and Pässler's investigations have been accepted, but only when this complication exists. For uncomplicated myocardial insufficiency the first principle is absolute rest. The digitalis group of drugs may be used; their efficacy depends largely upon the amount of myocardium affected. Predisposing causes should be controlled; in the case reported it was the pseudopertussis which was easily controlled by very small doses of codein. Stimulants will be required in most of the cases from alcohol to camphor or ether, according to the severity of the case. The convalescence must be especially guarded and, for the purpose of strengthening the myocardium, mechanical means are invaluable: Swedish movements, massage, vibratory methods. The diet is the same as in all myocardial affections. Laxatives that act violently should be avoided. Strychnia may be used for its general tonic effects; except in toxic doses it has no effect upon the heart nor upon the vasomotors. Nitroglycerin should never be used, unless there be anginous attacks; at all events, care must be taken to exclude the existence of vasomotor paralysis, when it does harm.

For the vasomotor paralysis Pässler has shown experimentally that digitalis produces only very transitory results, no effects upon the blood vessels; no effect is produced by alcohol or ether, possibly a bad one on the vasomotor centres; ergotin is without value; camphor produces good results, but the best are produced by caffein. Saline infusion produces temporary results and abdominal massage fairly good ones.

My own method, which I have used with good results in 2 cases, is as follows: hypodermic injection of adrenalin; this drug has an elective effect upon the blood vessels supplied by the splanchnic nerve. Its effects are very transitory; therefore, the injections have to be repeated every two hours. Saline transfusions are used in the beginning in order to keep up the heart's contractions. Ice-bags are put upon the abdomen; stimulating the splanchnic reflex. In one child I was able to bring the pulse down from 140 to 60 by the use of these alone. After the most violent symptoms have passed over, then caffein soda-salicylate is given, every two to four hours, alternating with the adrenalin, which is gradually dropped. The convalescence is treated as is indicated in the case reported.

COMPLICATIONS OF HERPES ZOSTER. (Journal of Neurol. and Psychiatry, London, 12: 152, May 1949). Whitty and Cooke describe 3 patients with herpes zoster, all of whom showed myelitis as a complication of the infection. Two had recurrent attacks of zoster in different skin zones over a period of two or three months, thus showing a difference from the usual behavior of the zoster virus, which confers lifelong immunity by a single attack. The authors consider the possibility that these were really cases of herpes simplex. The third patient had a zoster infection precipitated by injury to the fifth nerve. This case exemplifies a "symptomatic" zoster, secondary to peripheral trauma of the fifth cranial nerve. This is not uncommon after involvement of the first sensory neurone, especially at root level, by secondary neoplasm or tuberculous granuloma. Its cause is generally supposed to be the zoster virus, trauma being only a precipitating factor. Such a view is supported in this case by the sequel of midbrain encephalitis, inexplicable on a purely traumatic basis.—*Journal A.M.A.*

CLINICAL-PATHOLOGICAL CONFERENCE

WILLARD PARKER HOSPITAL, NEW YORK

Meeting held September 20, 1949

DR. PHILIP MOEN STIMSON, presiding.

MEASLES ENCEPHALITIS

DR. MARLOW presented Case No. 3025, a 7-year-old white female, who was admitted to this hospital on July 2, 1949, with a history of fever and listlessness which began six days prior to admission. She developed a conjunctivitis and cough, and four days before admission broke out in a generalized rash. She continued to be febrile and was put on sulfonamides, aspirin and cough medicine, two days before admission; penicillin on day of admission. On morning of day of admission patient had a series of convulsive seizures which were treated with phenobarbital, ether and injections. On admission, she was drowsy but responded to stimuli and had a negative neurological examination except for a right Babinski. Other findings: generalized lymphadenopathy, diffuse measle rash, enlarged, injected tonsils and systolic murmur at apex of heart. A spinal tap attempt was unsuccessful because of child's resistance.

The following day she was more alert but very irritable. She took fluids readily by mouth. Findings were unchanged except for stiffness of neck and sluggish deep, tendon reflexes. A spinal tap yielded opalescent fluid with a cell count of about 500 cells (65 per cent lymphs, 10 per cent red cells), a one plus Pandy and and a normal sugar. Patient lapsed into a semicomatose state and showed twitching of muscles of her extremities and face the next day. Her neck was rigid and Kernig positive. Babinski present, bilaterally. Knee jerks and abdominals depressed. She was given feedings by gavage and oxygen. She gradually lapsed into coma and showed deviation of pupils to the right, absence of reflexes in lower extremities and distention of the bladder. Her arms became spastic in flexion. Gamma globulin injection given.

On July 5, 1949, patient was seen by Dr. Appelbaum and aureomycin, 4 gm. daily, was begun. At this time she showed deviation of eyes to the right and skew deviation, absent reflexes, Biot type

breathing, in addition to signs noted before. That evening her temperature spiked to 107° F. and she had convulsive movements of upper extremities and deviation of eyes to the left. The convulsions ceased after ether and phenobarbital were given. Numerous rhonchi were present in both lungs.

The child's mental state remained unchanged and respirations were labored. Neurological signs remained largely the same and temperature elevations were only moderate. She was maintained on gavage and intravenous feedings. Pulse was out of proportion to temperature and on July 9, 1949, her liver was felt one finger below the costal margin. Left chest showed decreased breath sounds. On July 10, 1949, periorbital edema was noted. Amigen therapy begun. The following day her liver was two fingers below the costal margin and edema of face had increased. Intravenous fluids cut and mercuhydrin given. Lower extremities were flaccid but other neurological signs unchanged. On July 12, 1949, respirations ceased but heart beat was strong. Child was placed in respirator. Six hours later, patient became cyanotic and heartbeat ceased.

Dr. Dolgopol stated that it may be interesting to know that she spoke with Dr. Dolgin, who used to be our resident and who stated that the girl was over the measles and was in such good general condition that her mother telephoned him in the morning asking him if the child could leave her bed. However, he advised to keep her in bed until he visited her. In the late afternoon, the girl developed neurological symptoms.

Dr. Stimson asked if any one was present that had seen the patient in July when the patient was at this hospital.

Dr. Jurow stated that he had seen the patient.

Dr. Stimson asked if any one had anything further to contribue to this case. He also stated that this is about the most dreaded form of encephalitis.

Dr. Dolgopol showed the gross specimens. The brain weighed 1,360 grams and was congested and edematous, but showed no other gross changes. The lungs showed several small areas of atelectasis, but the left lung was almost completely atelectatic. The left pleural cavity contained about 20 cc. of clear yellowish fluid. On pressure droplets of thick creamy pus appeared on cut surface from the small bronchi of the left lung and of the atelectatic areas

of the right lung. The heart was somewhat enlarged. The myocardium was firm, but several hemorrhages were found within the interventricular septum. The valves showed no significant changes. The liver was enlarged and congested and weighed 900 grams. The spleen was small, but the kidneys were slightly enlarged.

DR. STIMSON asked Dr. Dolgopol if she could tell how old the hemorrhages in the myocardium were.

DR. DOLGOPOL stated that they were probably a day or two old. Dr. Dolgopol also stated that Dr. Silverberg had mentioned a new bedside electrocardiograph apparatus that could be run very easily and gives a direct reading. Perhaps, by the use of such a machine, we could get more electrocardiographic work done.

DR. GOLDSTEIN asked is it possible that the hemorrhage in the septum was the result of puncture with the needle during the administration of adrenal?

DR. DOLGOPOL. No. They could get into the right heart, but not into the muscles of the septum.

Microscopic Sections were projected. There was extensive loss of myelin throughout the brain, with maximal changes in the calcarine fissure of the occipital lobes and optic nerves. Areas of myelination were large and showed no definite perivascular pattern, apparently because of confluence of the foci. The motor area was least affected. The ganglion cells were only slightly damaged. This is the usual picture in measles encephalitis of more than a week's duration with the exception of extensive confluence of the foci. In the first two days there is usually no loss of myelin, but slight perivascular cuffing is observed in the gray and white matter.

A section from the heart in the area of hemorrhages showed Zenker's degeneration of myocardium and infiltration with polymorphonuclears and lymphocytes.

Sections from the lungs showed edema fluid and red blood cells in the alveolar lumen. The bronchi in the atelectatic areas contained mucus and cellular debris. There were no significant changes in other organs.

DR. GOLDSTEIN. What was the cause of the edema about the eyes?

DR. DOLGLPOL. The facial edema was not of renal origin as the kidneys showed no significant pathology.

DR. STIMSON. Many years have gone by but that it was customary at this hospital to see a list of deaths, 25-30. But in 1948, there was not a single death of scarlet fever. Here in August, we had 26 deaths at the hospital, but 19 of them were polio. The death rate has been very low at this hospital, even taking into consideration polio.

POLIOMYELITIS

DR. JUROW presented Case No. 3380, a 6-year-old white boy, admitted to Willard Parker Hospital on July 27, 1949, at 5:30 P. M. with a history of headache and anorexia on July 22, 1949. He felt well after this until July 25, 1949, when he again complained of headache, was feverish and ate little. He was seen by a private doctor the next day and penicillin tablets were prescribed. He was febrile, irritable, vomited all feedings and appeared continually to have much saliva in his mouth on the day of admission.

The patient appeared acutely ill, restless, pale and slightly cyanotic on admission. His breathing was rapid, irregular and shallow. There was a slight, left upper and lower facial weakness. His tongue deviated to the left and his soft palate was asymmetrical, the left side higher than the right. He was unable to swallow and had difficulty in spitting. His neck was supple, Brudzinski, negative, Kernig, questionably positive. Abdominal reflexes, absent. Deep tendon reflexes active and equal. Lungs were clear to percussion, and rhonchi were heard throughout both lung fields. Spinal tap showed 205 cells, 95 per cent lymphocytes, 5 per cent polys, Pandy, negative.

At about 8:00 P. M., while being put in oxygen tent, child's respiration and pulse suddenly stopped. Attempts at revival were unsuccessful.

DR. STIMSON. He had died when the artificial respiration was being applied. It is possible that the artificial respiration pulled a plug down.

DR. DOLGOPOL. Dr. Stimson, I wish to mention that there was no evidence of bronchial obstruction. Even microscopically, most of the bronchi were opened.

DR. STIMSON. Many people could not have seen the patient in those two and a half hours.

Dr. Seanor. I saw the patient immediately after death.

Dr. Dolgopol. It is seldom for us to see a tongue between the teeth of a polio patient. I want to ask if that was not the result of the artificial respiration that they pulled out the tongue,

Dr. Stimson. He had a partial paralysis of the tongue.

Dr. Jurow. The tongue was probably pulled forward in suctioning his pharynx.

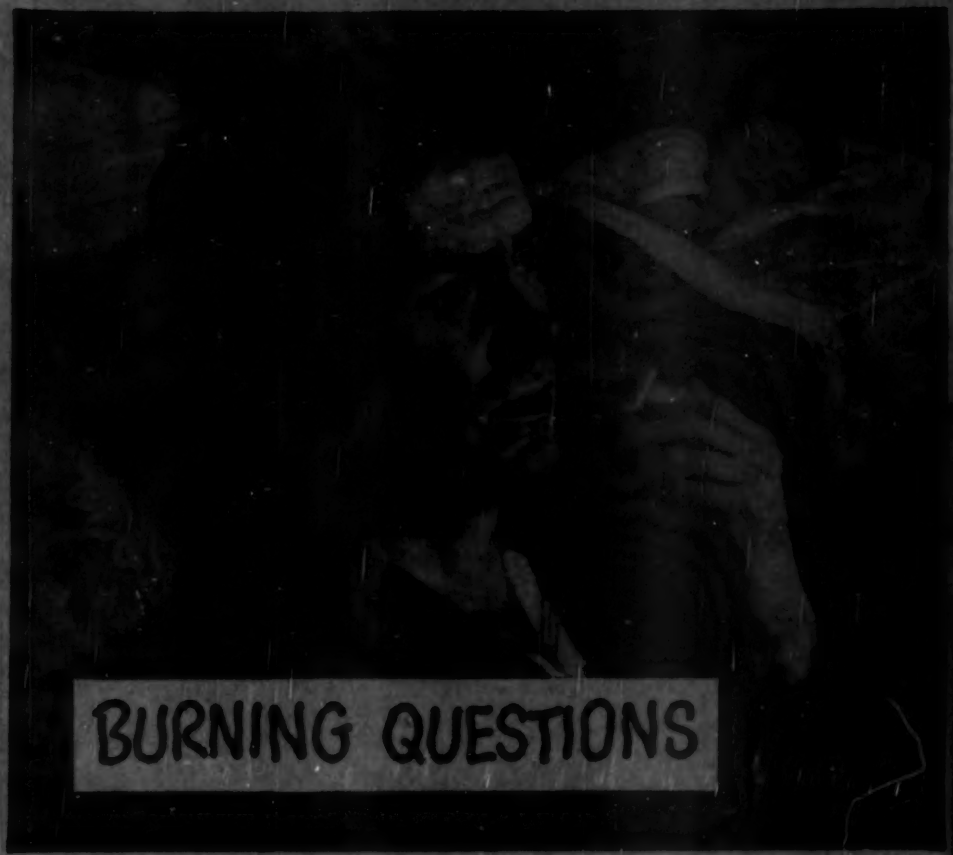
Autopsy Findings. Gross findings were presented by Dr. Dolgopol. The child's diaphragm was high and at the same time the liver was two finger-breadths above the costal margin which means it was markedly enlarged. The urinary bladder was quite distended, three finger-breadths above the pubis, which was quite high above the pubis for a child. There were many large yellowish lymph nodes in the mesentery. The pericardial cavities contained a few cubic centimeters of fluid. The heart showed large hemorrhages, larger than those seen in the measles case just shown. The pleural cavities were free from fluid. The posterior aspects of both lungs were dark-red in color but the tissue did not sink in water; this means that there was some air. The stomach showed a capillary congestion. Small intestines contained large Peyer's patches. The brain was large for a child of this age. The meninges were injected. No gross hemorrhages were seen in the spinal cord. This case did not show any clinical evidence of damage to the exremities, but there was definite microscopic damage to the cord, including the sacral cord. This, perhaps, could explain the retention of urine.

Microscopic Findings. The anterior aspect of the cord shows severe marked perivascular cuffing. Many glial nodules and diffuse gliosis is seen on both horns of the lumber region. There is congestion of the cerebrum. In the midbrain there is extensive perivascular cuffing and hemorrhages. The medulla showed practically a diffuse involvement, with loss of ganglion cells, perivascular cuffing, glial nodules, etc. There is practically no area that is not affected.

Dr. Stimson. There is not much edema there.

Dr. Dolgopol. This case is very interesting because there was a marked damage of the cord in the absence of clinical manifestations of cord damage.

Dr. Stimson. Neurosurgeons tell us that use of 10 per cent. glucose solutions intravenously helps in cerebral edema(?).



BURNING QUESTIONS

Late at night, some men do their hardest worrying. Each thought glows like the burning end of a cigarette. Chain-thoughts like:

"How am I doing my job? Have I reached my top? Are my best earning years numbered?

"And how will that affect my other job—as husband, father, family provider? Will I be able to do the things we've planned? What about college for the children? And our home—will I always be able to meet payments?"

Every man has to ask himself these questions. And not till he finds the right answers will the worry about the future cease.

One fundamental answer, of course, lies in a systematic plan of saving—one that builds soundly for the years ahead.

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There is the Payroll Savings Plan—an automatic system that tucks away a part of your earnings each payday into U. S. Savings Bonds. Bonds that pay you back four dollars for every three, after ten years.

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